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Inventor search

DATE: Tuesday, September 11, 2007

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☐	L3	(martin anne)[IN]	13
☐	L2	L1 and aurora-A	2
☐	L1	(prigent claude)[IN]	6

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NEWS 23	AUG 06	FSTA enhanced with new thesaurus edition
NEWS 24	AUG 13	CA/CAplus enhanced with additional kind codes for granted patents
NEWS 25	AUG 20	CA/CAplus enhanced with CAS indexing in pre-1907 records
NEWS 26	AUG 27	Full-text patent databases enhanced with predefined patent family display formats from INPADOCDB
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FILE LAST UPDATED: 10 Sep 2007 (20070910/ED)

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=> s (Aurora-2) or (Aur-2) or (STK-15) or (AIK) or (ARK1) or (AurA) or (AURA)
          4145 AURORA
           495 AURORAS
          4226 AURORA
                 (AURORA OR AURORAS)
       9285941 2
           113 AURORA-2
                 (AURORA(W)2)
           698 AUR
             1 AURS
           699 AUR
                 (AUR OR AURS)
       9285941 2
            12 AUR-2
                 (AUR(W)2)
           424 STK
            31 STKS
           452 STK
                 (STK OR STKS)
       1750137 15
             4 STK-15
                 (STK(W)15)
           130 AIK
             1 AIKS
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            130 AIK
                  (AIK OR AIKS)
            184 ARK1
            586 AURA
              7 AURAS
            593 AURA
                  (AURA OR AURAS)
            586 AURA
              7 AURAS
            593 AURA
                  (AURA OR AURAS)
L1         1027 (AURORA-2) OR (AUR-2) OR (STK-15) OR (AIK) OR (ARK1) OR (AURA)
                OR (AURA)

=> s L1 and (monoclonal)
         148722 MONOCLONAL
            543 MONOCLONALS
         148789 MONOCLONAL
                  (MONOCLONAL OR MONOCLONALS)
L2             8 L1 AND (MONOCLONAL)

=> duplicate remove L2
PROCESSING COMPLETED FOR L2
L3             8 DUPLICATE REMOVE L2 (0 DUPLICATES REMOVED)

=> d L3 bib abs 1-8

L3   ANSWER 1 OF 8  CAPLUS  COPYRIGHT 2007 ACS on STN
AN   2006:1158010  CAPLUS
DN   145:469962
TI   Monoclonal anti-MCM2 protein antibodies for diagnosis,
     prevention and treatment of cervical diseases
IN   Fischer, Timothy J.; Malinowski, Douglas P.; Taylor, Adriann J.
PA   Tripath Imaging, Inc., USA
SO   PCT Int. Appl., 59pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.          KIND   DATE        APPLICATION NO.        DATE
     ---------------     ----   --------    --------------------   --------
PI   WO 2006116442        A2    20061102    WO 2006-US15706        20060426
     WO 2006116442        A3    20070705
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
             CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
             GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR,
             KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX,
             MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE,
             SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC,
             VN, YU, ZA, ZM, ZW
         RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
             IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ,
             CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH,
             GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
             KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA
     US 2006252106        A1    20061109    US 2006-410272         20060424
     US 2007117167        A1    20070524    US 2006-643277         20061221
PRAI US 2005-675305P      P     20050427
     US 2005-718082P      P     20050916
     US 2006-410272       A3    20060424
AB   The invention provides a novel class of compds., pharmaceutical compns.
     comprising such compds. and methods of using such compds. to treat or
     prevent diseases or disorders associated with abnormal or deregulated kinase
     activity, particularly diseases or disorders that involve abnormal
```

activation of AIk, AbI, BRK, BIk, BMX, CSK, c-Src, c-Raf, EGFR, Fes, FGFR3, Fms, Fyn, IGF-IR, IR, IKKα, IKKβ, JAK2, JAK3, KDR, Lck, Met, p70S6k, Ros, Rskl, SAPK2α, SAPK2β, SAPK3, SIK, Tie2, TrkB and/or WNK3 kinases. The disease is a early stage HPV infection or cervical disease such as cervical carcinoma and mild dysplasia. The antibodies are specific to epitopes of MCM2 protein (Minichromosome maintenance protein 2).

L3 ANSWER 2 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:757393 CAPLUS
DN 139:271089
TI Phosphoinositide 3-kinase mediated inhibition of GPCRs
IN Rockman, Howard A.; Naga, Prasad Sathyamangla V.; Laporte, Stephane A.; Barak, Larry S.; Caron, Marc G.
PA USA
SO U.S. Pat. Appl. Publ., 71 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2003182669	A1	20030925	US 2002-101235	20020319
	WO 2003088924	A2	20031030	WO 2003-US8208	20030318
	WO 2003088924	A3	20051229		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

	AU 2003253586	A1	20031103	AU 2003-253586	20030318
	US 2006026702	A1	20060202	US 2004-902137	20040730
PRAI	US 2002-101235	A	20020319		
	WO 2003-US8208	W	20030318		

AB The present invention relates to compds. that alter G protein-coupled receptor (GPCR) internalization and new methods for their identification. Compds. of this invention include modified phosphoinositide 3-kinase (PI3K), modified HEAT domain, modified β-adrenergic receptor kinase 1 (β ARK1), as well as other peptides or small mols. that alter GPCR internalization. The present invention also includes the use of such compds. to treat GPCR-related diseases, such as cardiovascular disease, heart failure, asthma, nephrogenic diabetes insipidus, or hypertension.

L3 ANSWER 3 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:488124 CAPLUS
DN 137:59517
TI Human AURORA-1 and AURORA-2 kinases, cDNA and amino acid sequences, and recombinant production
IN Plowman, Gregory; Mossie, Kevin
PA Sugen, Inc., USA
SO U.S. Pat. Appl. Publ., 43 pp., Cont.-in-part of U.S. Ser. No. 5,268, abandoned.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 4

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

PI US 2002081578 A1 20020627 US 1998-12135 19980122
US 6716575 B2 20040406
CN 1205740 A 19990120 CN 1996-199101 19961125
US 5962312 A 19991005 US 1996-755728 19961125
EP 1655369 A1 20060510 EP 2005-23434 19961125
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI
CA 2318352 A1 19990729 CA 1999-2318352 19990121
WO 9937788 A2 19990729 WO 1999-US1283 19990121
WO 9937788 A3 19990930
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
AU 9925605 A 19990809 AU 1999-25605 19990121
EP 1051500 A2 20001115 EP 1999-905450 19990121
EP 1051500 B1 20050817
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI
JP 2002508937 T 20020326 JP 2000-528695 19990121
AT 302278 T 20050915 AT 1999-905450 19990121
ES 2247783 T3 20060301 ES 1999-905450 19990121
US 6207401 B1 20010327 US 1999-283011 19990331
US 2005002938 A1 20050106 US 2001-784332 20010216
US 6841579 B2 20050111
US 2004265852 A1 20041230 US 2003-734126 20031215
US 7119174 B2 20061010
PRAI US 1995-8809P P 19951218
US 1996-23943P P 19960814
US 1996-755728 A2 19961125
US 1998-5268 B2 19980109
EP 1996-940870 A3 19961125
US 1998-12135 A 19980122
WO 1999-US1283 W 19990121
US 1999-283011 A3 19990331
US 2001-784332 A3 20010216

AB The invention provides protein and cDNA sequences for human AURORA-1 (AUR1) and/or AURORA-2 (AUR2), which are members of serine/threonine kinase family containing short N-terminal extensions. AUR1 mRNA has been shown to be broadly expressed in rapidly dividing cells, derived from both normal and tumor tissues. AUR2 mRNA, however, has been shown to be expressed in a more restricted pattern being low or absent in most normal tissues and abundant in only a subset of tumor-derived cell lines. The invention also demonstrated that AUR1 and AUR2 kinases were able to phosphorylate myelin basic protein. The invention further discussed the possible involvement of AUR1 and AUR2 kinases in cancer and/or other signal transduction disorders, and the possible biol., diagnostic and/or therapeutic uses of these kinases. The AUR1 and AUR2 genes are mapped to chromosome 17p13.1 and 20q13.2 resp. Methods for treatment, diagnosis, and screening are provided for AUR1 and/or AUR2 related diseases or conditions characterized by an abnormal interaction between a AUR1 and/or AUR2 polypeptide and a AUR1 and/or AUR2 binding partner.

RE.CNT 182 THERE ARE 182 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 4 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:636823 CAPLUS
DN 137:165497
TI Method and kit for assaying protein phosphorylation enzyme activity, and

antibody used for assay
IN Taji, Shingo; Tamai, Katsuyuki; Kobayashi, Toshiko
PA Medical and Biological Laboratories Co., Ltd., Japan
SO Jpn. Kokai Tokkyo Koho, 29 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 2002236125	A	20020823	JP 2001-29774	20010206
PRAI JP 2001-29774		20010206		

AB A method and a kit are provided for assaying a human Aurora2 protein phosphorylation enzyme activity using an antibody capable of specifically recognizing and binding with the substrate phosphorylated with human Aurora2 protein phosphorylation enzyme. An antibody used for assaying a human Aurora2 protein phosphorylation enzyme activity is also provided. A method is also provided for screening a compound which inhibits or promotes the human Aurora2 protein phosphorylation enzyme activity. The phosphorylation activity of human Aurora2 protein phosphorylation enzyme is assayed by immunol. measuring the phosphorylation of its substrate using an antibody capable of specifically recognizing and binding with the substrate phosphorylated with human Aurora 2 protein phosphorylation enzyme (e.g., human Lats2 protein).

L3 ANSWER 5 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:430919 CAPLUS
DN 137:227360
TI Comparison of two aquatic alphaviruses, salmon pancreas disease virus and sleeping disease virus, by using genome sequence analysis, monoclonal reactivity, and cross-infection
AU Weston, Jonathan; Villoing, Stephane; Bremont, Michel; Castric, Jeanette; Pfeffer, Martin; Jewhurst, Victoria; McLoughlin, Marian; Roedseth, Odd Magne; Christie, Karen Elina; Koumans, Joseph; Todd, Daniel
CS Department of Veterinary Sciences, The Queen's University of Belfast, Belfast, BT4 3SD, UK
SO Journal of Virology (2002), 76(12), 6155-6163
CODEN: JOVIAM; ISSN: 0022-538X
PB American Society for Microbiology
DT Journal
LA English
AB Cell culture isolates of salmon pancreas disease virus (SPDV) of farmed Atlantic salmon and sleeping disease virus (SDV) of rainbow trout were compared. Excluding the poly(A) tracts, the genomic nucleotide sequences of SPDV and SDV RNAs include 11,919 and 11,900 nucleotides, resp. Phylogenetic anal. places SPDV and SDV between the New World viruses of Venezuelan equine encephalitis virus and Eastern equine encephalitis virus and the Old World viruses of Aura virus and Sindbis virus. When compared to each other, SPDV and SDV show 91.1% nucleotide sequence identity over their complete genomes, with 95 and 93.6% amino acid identities over their nonstructural and structural proteins, resp. Notable differences between the two viruses include a 24-nucleotide insertion in the C terminus of nsP3 protein of SPDV and amino acid sequence variation at the C termini of the capsid and E1 proteins. Exptl. infections of Atlantic salmon and rainbow trout with SPDV and SDV confirmed that the disease lesions induced by SPDV and SDV were similar in nature. Although infections with SPDV and SDV produced similar levels of histopathol. in rainbow trout, SDV induced significantly less severe lesions in salmon than did SPDV. Virus neutralization tests performed with sera from exptl. infected salmon indicated that SPDV and SDV belonged to the same serotype; however, antigenic variation was detected among SDV and geog. different SPDV isolates by using monoclonal antibodies. Although SPDV and SDV exhibit minor biol. differences, we conclude on the basis of the close genetic similarity that SPDV and SDV

are closely related isolates of the same virus species for which the name Salmonid alphavirus is proposed.
RE.CNT 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:195040 CAPLUS
DN 137:92550
TI Expression of NOS-2, COX-2 and Th1/Th2 cytokines in migraine
AU Martelletti, Paolo; Zicari, Alessandra; Realacci, Massimo; Fiore, Giuseppe; De Filippis, Sergio; Stirparo, Giuseppe; Denora, Paola; Solimeo, Maria Donata; Rinaldi, Cristina; Morrone, Stefania; Giacovazzo, Mario
CS Internal Medicine, Headache Centre, Sant'Andrea Hospital, 2nd School of Medicine, La Sapienza University of Rome, Rome, I-00189, Italy
SO Journal of Headache and Pain (2001), 2(Suppl. 1), S51-S56
CODEN: JHPOAT; ISSN: 1129-2369
PB Springer-Verlag Italia Srl
DT Journal
LA English
AB Nitric oxide (NO) probably plays an important role in the pathogenesis of migraine without aura (MWA). As the activation of NO-ergic cascade has been shown to be closely linked to cyclooxygenase pathway and to cause some differences in peripheral blood lymphocyte populations, we investigated if the Th1/Th2 balance in peripheral blood of MWA patients was affected in comparison to controls. Twenty-six MWA patients and 10 healthy controls (C) were enrolled in this study. Blood samples were taken at baseline (T0) and during an induced migraine attack (T1). Total RNA from human peripheral blood lymphocytes (PBLs) was isolated and reverse-transcribed to prepare complementary DNA. COX-2, NOS-2 and β-actin were amplified using PCR. PBLs from patients and controls were stimulated with phorbol 12-myristate 13-acetate plus ionomycin in the presence of brefeldin A. Cells were surface-stained with fluorochrome-conjugated anti-CD3 and anti-CD8 monoclonal antibodies (mAbs) and intracellularly stained with fluorochrome-conjugated anti-IFN-γ or anti-IL-4 mAbs. The level of cytokine expression was analyzed by gating on the CD4+ lymphocyte subset. Th1 and Th2 type cytokines (IFN-γ, IL-2, IL-4) were directly assayed in serum by ELISA. Preliminary results indicate that NOS-2 was upregulated in MWA patients at basal time if compared to controls, whereas after NOD administration NOS-2 was significantly decreased. COX-2 was upregulated in MWA patients at basal time and it had an opposite trend after NOD administration. The homeostatic Th1/Th2 balance defined by the IFN-γ or IL-4 cytokine expression was unchanged in MWA patients in comparison to controls, and NOD administration did not affect that pattern. The cell activation machinery was not altered after mitogenic activation, as shown by CD69 expression level. Cytokine serum levels showed no significant changes in all studied situations. This study confirms the relevance of the NOS/COX system in MWA but, in contrast with previous studies, excludes their effect and activation on peripheral cytokine production More sophisticated exptl. models are needed to investigate the ability of NOS/COX to activate migraine pain.
RE.CNT 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 7 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1995:941486 CAPLUS
DN 124:25278
TI Analysis of the hemagglutination activity domains of the Venezuelan equine encephalomyelitis and eastern equine encephalomyelitis viruses
AU Razumov, I. A.; Khusainova, A. D.; Agapov, E. V.; Gajdamovich, S. Ya.; Pereboev, A. V.; Kolykhalov, A. A.; Netesov, S. V.; Loktev, V. B.
CS State Research Center Virology and Biotechnology "Vector", Institute Molecular Biology, Koltsovo, 633159, Russia
SO Intervirology (1995), Volume Date 1994, 37(6), 356-60

CODEN: IVRYAK; ISSN: 0300-5526
PB Karger
DT Journal
LA English
AB The hemagglutination (HA) domains of the Venezuelan equine encephalomyelitis (VEE) and the eastern equine encephalomyelitis (EEE) viruses providing the interaction of virons and red blood cells were studied with the use of a panel of 17 hemagglutination inhibition (HI) monoclonal antibodies (MAbs). A highly conserved domain (C domain) forming alphavirus-group-reactive MAbs inhibited in the E2 protein of the VEE and EEE viruses. These MAbs inhibited HA of the western equine encephalomyelitis, Semliki Forest, Sindbis, Getah, Aura, Chikungunya and Pixuna viruses. The involvement of amino acid residues 59 and 232 in the formation of the C region was demonstrated by sequencing the gene encoding the E2 protein of three escape variants of the VEE virus.

L3 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1994:241981 CAPLUS
DN 120:241981
TI Molecular studies of alphavirus immunogenicity
AU Strauss, J. H.
CS California Inst. Tech., Pasadena, CA, USA
SO Report (1992), Order No. AD-A261546, 46 pp. Avail.: NTIS
From: Gov. Rep. Announce. Index (U. S.) 1993, 93(14), Abstr. No. 341,843
DT Report
LA English
AB The alphaviruses consist of a group of 26 closely related viruses. Many of these viruses can cause disease in man, characterized by encephalitis, polyarthritis, fever or rash, depending upon the virus. In the 2.5 yr of research supported under this contract the authors have mapped antigenic epitopes in the structural glycoproteins of alphaviruses that lead to neutralization of virus infectivity upon reaction with an antibody, and have determined the sequence relationships of a number of Sindbis-like alphaviruses to one another and to other alphaviruses. The authors found that a domain of glycoprotein E2 of alphaviruses, between residues of 170 and 220, was an important region for binding of monoclonal antibodies that neutralize virus infectivity, making it critical importance for the immune response required for protection from infection by the virus.. In the determination of the relationships of alphaviruses to one another, the authors have determined complete or partial sequences of 8 different alphavirus RNAs. These include Ockelbo virus, a virus causing epidemic polyarthritis in northern Europe, strains of Sindbis virus from Africa, Inda, Australia and New Zealand arid Aura virus from South America...

=> s ((Aurora-A) or STK-15) and monoclonal
4145 AURORA
495 AURORAS
4226 AURORA
(AURORA OR AURORAS)
21285116 A
501 AURORA-A
(AURORA(W)A)
424 STK
31 STKS
452 STK
(STK OR STKS)
1750137 15
4 STK-15
(STK(W)15)
148722 MONOCLONAL

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          543 MONOCLONALS
       148789 MONOCLONAL
                (MONOCLONAL OR MONOCLONALS)
L4          12 ((AURORA-A) OR STK-15) AND MONOCLONAL

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PROCESSING COMPLETED FOR L4
L5          12 DUPLICATE REMOVE L4 (0 DUPLICATES REMOVED)

=> d L5 bib abs 1-12

L5   ANSWER 1 OF 12  CAPLUS  COPYRIGHT 2007 ACS on STN
AN   2007:488925  CAPLUS
DN   147:7139
TI   Subcellular localization of the spindle proteins Aurora
     A, Mad2, and BUBR1 assessed by immunohistochemistry
AU   Burum-Auensen, Espen; De Angelis, Paula M.; Schjoelberg, Aasa R.; Kravik,
     Katherine L.; Aure, Marit; Clausen, Ole Petter F.
CS   The Pathology Clinic, Rikshospitalet-Radiumhospitalet Medical Center,
     Faculty of Medicine, University of Oslo, Oslo, Norway
SO   Journal of Histochemistry and Cytochemistry (2007), 55(5), 477-486
     CODEN: JHCYAS; ISSN: 0022-1554
PB   Histochemical Society, Inc.
DT   Journal
LA   English
AB   The spindle checkpoint, the primary mechanism to ensure that two daughter
     cells receive the same amount of DNA, is compromised in many malignant
     tumors and has been implicated as a contributor to aneuploidy and
     carcinogenesis.  The extent of expression and subcellular localization of
     the spindle proteins Aurora A, Mad2, and BUBR1 varies
     considerably in different immunohistochem. (IHC) reports from archival
     tumor tissues.  Given the conflicting reports in the literature about the
     localization of these proteins, we examined the subcellular localization of
     Aurora kinase A, Mad2, and BUBR1 in normal and cancerous human tissues by
     IHC.  In normal tissues, Aurora A was mainly localized
     to the nucleus when monoclonal or purified polyclonal antibodies
     were used, and Mad2 was localized to the nucleus, whereas BUBR1 was
     localized to the cytoplasm.  In malignant tissues, Aurora
     A showed addnl. staining in the cytoplasm in the majority of
     tumors analyzed.  Furthermore, BUBR1 was also localized to both the
     nucleus and cytoplasm in a significant fraction of tumors.  Subcellular
     localization of Mad2 was similar in normal and malignant tissues.  Thus,
     the validity of some earlier IHC studies of Aurora A,
     Mad2, and BUBR1 should be reconsidered, indicating that high-quality
     antibodies and a high-alkaline antigen-retrieval technique are required to
     achieve optimal results.  We conclude that the subcellular localizations
     of these spindle proteins are different, although they have overlapping
     biol. functions, and that Aurora A and BUBR1 undergo a
     shift in the subcellular localization during malignant transformation.
RE.CNT  44    THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD
              ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5   ANSWER 2 OF 12  CAPLUS  COPYRIGHT 2007 ACS on STN
AN   2006:1279840  CAPLUS
DN   146:45539
TI   Preparation of aminopyridine derivatives as selective Aurora-
     A inhibitors for treatment of cancer
IN   Kato, Tetsuya; Kawanishi, Nobuhiko; Mita, Takashi; Ohkubo, Mitsuru;
     Shimomura, Toshiyasu
PA   Banyu Pharmaceutical Co., Ltd., Japan
SO   PCT Int. Appl., 151pp.
     CODEN: PIXXD2
DT   Patent
LA   Japanese
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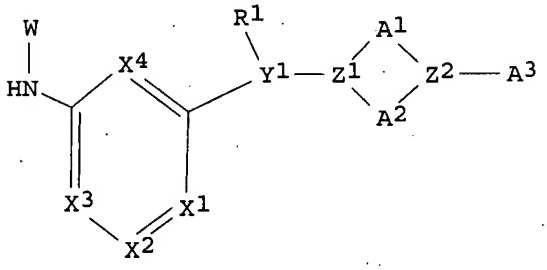
FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2006129842	A1	20061207	WO 2006-JP311179	20060530
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	WO 2006046734	A2	20060504	WO 2005-JP19957	20051025
	WO 2006046734	A3	20060921		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	US 2006106029	A1	20060518	US 2005-258447	20051025
PRAI	JP 2005-161156	A	20050601		
	WO 2005-JP19957	A	20051025		
	JP 2004-315152	A	20041029		
	US 2005-692537P	P	20050621		

OS MARPAT 146:45539

GI



I

AB The title compds. I [A1 is (RbjCRbj')m2; A2 is (RaiCRai')m1; A3 is (Y2Rc)n1CO(Y3Rd)n2R; m1 and m2 each is 1, 2, or 3; n1 and n2 each is 0 or 1; i is an integer of 1 to m1; j is an integer of 1 to m2; R is optionally substituted aryl, heteroaryl, or cycloalkyl; Rai and Rai' each is hydrogen, alkyl; Rbj and Rbj' each is hydrogen, alkyl; Rc, Rd, and R1 each is hydrogen, alkyl; X1 is CH, CX1a, N; X1a is (un)substituted alkyl; X2 is CH, N, etc.; X3 is CH, CX3a, N; X3a is (un)substituted alkyl; X4 is CH or N; Y1, Y2, and Y3 are the same or different and each is CH or N; Z1 and Z2 are the same or different and each is CH or N; and W is a 5-membered aromatic heterocycle, e.g., pyrazole or thiazole] are prepared Thus, (5-bromothiazol-2-yl)-(6-(4-benzoylpiperazin-1-ylmethyl)pyridin-2-yl)amine

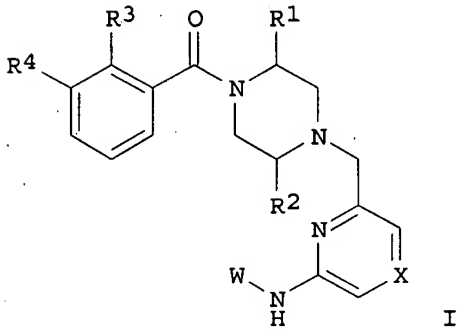
was prepared in a multistep process from 2-aminothiazole and 2,6-dichloropyridine. Compds. of this invention showed IC50 values of 0.36 nM to 110 nM against Aurora-A; they showed IC50 values of 47 nM to 28000 nM against Aurora-B.

RE.CNT 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 3 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:411981 CAPLUS
DN 144:450734
TI Preparation of novel aminopyridines having Aurora A selective inhibitory action
IN Ohkubo, Mitsuru; Kato, Tetsuya; Kawanishi, Nobuhiko; Mita, Takashi; Shimomura, Toshiyasu
PA Banyu Pharmaceutical Co., Ltd., Japan
SO PCT Int. Appl., 49 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 3

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2006046735	A1	20060504	WO 2005-JP19958	20051025
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
US 2006106029	A1	20060518	US 2005-258447	20051025
EP 1828165	A1	20070905	EP 2005-799006	20051025
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, HR				
PRAI JP 2004-315152	A	20041029		
JP 2005-161156	A	20050601		
US 2005-692537P	P	20050621		
WO 2005-JP19958	W	20051025		

OS MARPAT 144:450734
GI



I

AB The title compds. I [R1, R2 = H, lower alkyl, or alternatively R1 and R2 are combined together to form CH2; R3 = halo; R4 = halo or Me substituted

with 1-3 halogen atoms; X = CH, N; W = (un)substituted thiazolyl, pyrazolyl, thiadiazolyl; with the proviso] which are Aurora A selective inhibitors useful in combination therapy of cancer, were prepared E.g., a multi-step synthesis of I [R1, R2 = H; R3 = F; R4 = Cl; X = CH; W = 2-thiazolyl], starting from (6-bromopyridin-2-yl)methanol, was given (no characterization data provided for intermediates). The compds. I exhibit excellent Aurora A selective activity (biol. data were provided for exemplified compds. I). Pharmaceutical compns. comprising the compound I alone or in combination with other antitumor agent(s) were disclosed.

RE.CNT 24 THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 4 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:412038 CAPLUS
DN 144:450735
TI Preparation of novel aminopyridine derivatives having selective Aurora-A protein kinase inhibitory effect
IN Ohkubo, Mitsuru; Kato, Tetsuya; Kawanishi, Nobuhiko; Mita, Takashi; Shimomura, Toshiyasu
PA Banyu Pharmaceutical Co., Ltd., Japan
SO PCT Int. Appl., 148 pp.
CODEN: PIXXD2
DT Patent
LA Japanese
FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2006046734	A2	20060504	WO 2005-JP19957	20051025
	WO 2006046734	A3	20060921		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	AU 2005297848	A1	20060504	AU 2005-297848	20051025
	US 2006106029	A1	20060518	US 2005-258447	20051025
	EP 1813609	A2	20070801	EP 2005-799135	20051025
	R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, HR				
	WO 2006129842	A1	20061207	WO 2006-JP311179	20060530
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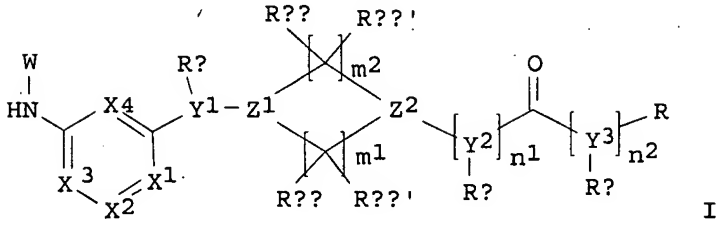
	RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	IN 2007DN03927	A	20070831	IN 2007-DN3927	20070525
PRAI	JP 2004-315152	A	20041029		
	JP 2005-161156	A	20050601		
	US 2005-692537P	P	20050621		

WO 2005-JP19957 W 20051025
OS MARPAT 144:450735
GI



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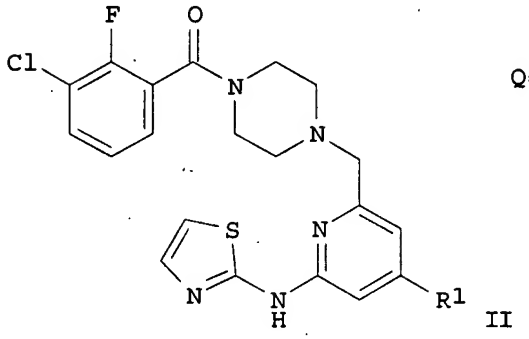
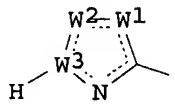


Q= 

II

AB The title compds. (I) or pharmaceutically acceptable salts or ester thereof [wherein m1, m2 = 1, 2, 3; n1, n2 = 0, 1; i = an integer of from 1 to m1; j = an integer of from 1 to m2; R = (un)substituted aryl, heteroaryl or cycloalkyl; Rai, Rai', Rbj, Rbj', Rc, Rd, Re = H, lower alkyl; X1 = CH, CX1a, N; wherein X1a = (un)substituted lower alkyl; X2 = CH, N; X3 = CH, N, CX3a; wherein X3a = (un)substituted lower alkyl; X4 = CH, N; 1 or 2 of X1-X4 is N ; Y1, Y2, Y3 = CH, N; Z1, Z2 = CH, N; W = a 5-membered aromatic heterocycle of formula Q including pyrazole or thiazole; wherein W1 = CH, N, NH, O, S; W2 = CH, CW2a, N, NW2b, O, S; wherein W2a, W2b = H, halo, cyano, C1-2 alkyl, C3-5 cycloalkyl, 1 or 2 halo-substituted C1-2 alkyl] are prepared These compds. are selective inhibitors of Aurora-A protein kinase over Aurora-B protein kinase and exhibit synergistic anticancer activity in combination with other anticancer agents. An anticancer agent containing the compound I, and the combined use of the above anticancer agent with another anticancer agent are also disclosed. Thus, a mixture of 2.70 g 6-chloromethyl-N-(thiazol-2-yl)pyridin-2-amine, 4.00 g 1-(3-chloro-2-fluorobenzoyl)piperazine, and 6.25 mL N,N-diisopropylethylamine, and 30 mL DMF was stirred at 90° for 2 h to give, after workup and silica gel chromatog., 6-[(4-(3-chloro-2-fluorobenzoyl)piperazin-1-yl)methyl]-N-thiazol-2-ylpyridin-2-amine (II; R = H). II (R = H) and II (R = 2-methyl-2H-tetrazol-5-yl) showed IC50 of 0.67 and 0.32 nM against Aurora-A protein kinase, resp., and 440 and 190 nM against Aurora-B protein kinase, resp. They showed IC50 of 11.00 and 0.21 μM against human cervical cancer cell (HeLa S3), resp., and also showed synergistic antiproliferative activity against HeLa S3 cells in combination with paclitaxel.

L5 ANSWER 5 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:166709 CAPLUS
DN 144:233067
TI 2-Amidothiazole-based compounds as inhibitors of ATP-utilizing enzymes,

their preparation, pharmaceutical compositions, and use in therapy

IN Dickson, John K., Jr.; Hodge, Carl Nicholas; Mendoza, Jose Serafin; Chen, Ke

PA Amphora Discovery Corporation, USA

SO PCT Int. Appl., 141 pp.
CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2006020767	A2	20060223	WO 2005-US28549	20050811
	WO 2006020767	A3	20061109		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	AU 2005272815	A1	20060223	AU 2005-272815	20050811
	CA 2575466	A1	20060223	CA 2005-2575466	20050811
	US 2006052416	A1	20060309	US 2005-202927	20050811
	EP 1781287	A2	20070509	EP 2005-803385	20050811
	R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, YU				
	IN 2007DN00668	A	20070803	IN 2007-DN668	20070124
PRAI	US 2004-601266P	P	20040813		
	US 2004-608834P	P	20040910		
	WO 2005-US28549	W	20050811		

OS MARPAT 144:233067

GI

* STRUCTURE DIAGRAM TOO LARGE FOR DISPLAY - AVAILABLE VIA OFFLINE PRINT *

AB The invention relates to 2-amidothiazole compds. of formula I, which are inhibitors of ATP-utilizing enzymes, such as synthetases, ligases, and kinases. In compds. I, R is OH, alkoxy, (un)substituted amino, (un)substituted cycloalkyl, (un)substituted aryl, or (un)substituted heteroaryl; L is a bond, carbonyl, -NHC(O)-, (un)substituted C1-4 alkylene, C1-4 alkylene-NHC(O)-, or C1-4 alkylene-C(O)-; W is selected from H, halo, (un)substituted alkyl, (un)substituted cycloalkyl, (un)substituted heterocyclyl, (un)substituted aryl, and (un)substituted heteroaryl; Q is (un)substituted alkyl, (un)substituted cycloalkyl, (un)substituted heterocyclyl, (un)substituted aryl, or (un)substituted heteroaryl; and Z is (un)substituted alkyl; with several provisos. The invention also relates to the preparation of I, pharmaceutical compns. comprising a therapeutically effective amount of a compound I, optionally one or more addnl. therapeutic agents, and at least one pharmaceutically acceptable vehicle, as well as to the use of the compns. for the treatment of conditions associated with ATP-utilizing enzymes. Addition of tert-Bu 3-aminopropanoate (β-alaninate) to N-Fmoc-isothiocyanate followed by deprotection gave thiourea II, which cyclized with 2-(bromoacetyl)benzofuran to give aminothiazole III. Amine III was acylated with thiophene-2-carbonyl chloride followed by ester cleavage and

amidation with nipecotamide (piperidine-3-carboxamide), resulting in the formation of amidothiazole IV. Some compds. of the invention express IC50 values of less than 30 μM in cellular proliferation assays and some express EC50 values of less than 30 μM in an assay for the induction of apoptosis in target cells.

L5 ANSWER 6 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:99983 CAPLUS
DN 144:184708
TI Use of K-252a and kinase inhibitors for the prevention or treatment of HMGB1-associated pathologies
IN Fumero, Silvano; Pilato, Francesco, P.; Barone, Domenico; Bertarione, Rava, Rossa, Luisa; Mainero, Valentina; Traversa, Silvio
PA Creabilis Therapeutics S.p.A., Italy; Bio3research Srl
SO PCT Int. Appl., 63 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2006010628	A1	20060202	WO 2005-EP8258	20050729
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
AU 2005266447	A1	20060202	AU 2005-266447	20050729
CA 2575272	A1	20060202	CA 2005-2575272	20050729
EP 1771178	A1	20070411	EP 2005-778429	20050729
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR				
PRAI US 2004-591880P	P	20040729		
US 2005-647007P	P	20050127		
WO 2005-EP8258	W	20050729		

AB The present invention relates to the use of K-252a, a physiol. active substance produced by microorganisms, and/or a kinase inhibitor and of its salts or synthetic and/or chemical modified derivs. for the prevention or treatment of HMGB1-associated pathologies. More particularly, the present invention relates to the use of K-252a for the prevention or treatment of restenosis.
RE.CNT 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 7 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:1309859 CAPLUS
DN 146:179238
TI Phospho-regulation of human protein kinase Aurora-A: Analysis using anti-phospho-Thr288 monoclonal antibodies
AU Ohashi, S.; Sakashita, G.; Ban, R.; Nagasawa, M.; Matsuzaki, H.; Murata, Y.; Taniguchi, H.; Shima, H.; Furukawa, K.; Urano, T.
CS Department of Biochemistry II, Nagoya University Graduate School of Medicine, Showa-ku, Nagoya, Japan
SO Oncogene (2006), 25(59), 7691-7702
CODEN: ONCNES; ISSN: 0950-9232
PB Nature Publishing Group
DT Journal

LA English
AB Mammalian Aurora-A is related to a serine/threonine protein kinase that was originally identified by its close homol. with Saccharomyces cerevisiae Ipl1p and Drosophila melanogaster aurora that are key regulators in the orchestration of mitotic events. The protein level of Aurora-A, its peak kinase activity during mitosis, and its activation have been attributed to phosphorylation. Here we show that this enzyme is an arginine-directed kinase and define its substrate specificity. We also found that Thr288 within the activation loop is a critical residue for activating phosphorylation events in vitro and that it is spatiotemporally restricted to a brief window at mitosis on duplicated centrosomes and on spindle microtubules proximal to the poles in vivo. Immunodepletion assays indicated that an upstream kinase(s) of Aurora-A might exist in mammalian cells in addition to autophosphorylation. Furthermore, human activated Aurora-A forms complexes with the neg. regulator protein serine/threonine phosphatase type 1 (PP1) that was neg. phosphorylated on Thr320. Interestingly, phospho-specific Aurora-A monoclonal antibodies restrain Aurora-A kinase activity in vitro, providing further therapeutic avenues to explore.
RE.CNT 79 THERE ARE 79 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 8 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:33225 CAPLUS
DN 142:112460
TI Monoclonal antibodies to fragment of human mitotic kinase Aurora-A phosphorylated at threonine 288, preparation, and use in cancer therapy
IN Urano, Takeshi; Furukawa, Koichi
PA Farma Design Inc., Japan
SO Jpn. Kokai Tokkyo Koho, 16 pp.
CODEN: JKXXAF
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI JP 2005006532	A	20050113	JP 2003-172730	20030618
PRAI JP 2003-172730		20030618		

AB This invention relates to antibodies, particularly, monoclonal antibodies, against human mitotic kinase Aurora-A (Aur-A) phosphorylated at threonine 288 (Thr-288), production in hybridoma, and use in treatment of diseases associated with Aur-A (over)expression, notably cancer. Monoclonal antibodies (mAbs) were raised against human Thr-288 phosphorylated Aur-A fragment. The mAbs were able to inhibit activation of Aur-A via phosphorylation of Thr-288.

L5 ANSWER 9 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:1059119 CAPLUS
DN 142:32932
TI Combination therapy for cancer and other proliferative disorders
IN Blatt, Lawrence M.; Seiwert, Scott D.; Ozes, Osman N.
PA Intermune, Inc., USA
SO PCT Int. Appl., 635 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004105684	A2	20041209	WO 2004-US15346	20040513
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,				

GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE,
SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE,
SN, TD, TG

PRAI US 2003-471841P P 20030516
US 2003-485474P P 20030708
US 2003-511259P P 20031014
US 2003-511280P P 20031014
US 2003-511415P P 20031014
US 2003-514173P P 20031024
US 2004-561940P P 20040413

AB The invention provides methods of treating proliferative disorders, including angiogenesis-mediated disorders, cancer, and fibrotic disorders. In some embodiments, the methods involve administering a Type II interferon receptor agonist and a Type I interferon receptor agonist. In other embodiments, the methods involve administering a Type II interferon receptor agonist, a stress-activated protein kinase (SAPK) inhibitor, and a third therapeutic agent. In other embodiments, the methods involve administering a Type II interferon receptor agonist and a vascular endothelial growth factor (VEGF) antagonist. In other embodiments, the methods involve administering a VEGF antagonist and a SAPK inhibitor. The invention further provides methods of treating fibrotic disorders. In some embodiments, the methods involve administering a Type I interferon receptor agonist, a Type II interferon receptor agonist; and a tumor necrosis factor (TNF) antagonist. In other embodiments, the methods involve administering a Type II interferon receptor agonist and a TNF antagonist. In other embodiments, the methods involve administering pirfenidone or a pirfenidone analog and a TNF antagonist. In other embodiments, the methods involve administering a Type II interferon receptor agonist and a transforming growth factor-β (TGF-β) antagonist. In other embodiments, the methods involve administering a SAPK inhibitor alone or in combination with a Type II interferon receptor agonist. In other embodiments, the methods involve administering N-acetyl cysteine (NAC) and a SAPK inhibitor. In other embodiments, the methods involve administering NAC and a Type II interferon receptor agonist.

L5 ANSWER 10 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:1079852 CAPLUS
DN 142:69142
TI Protein phosphatase CDC25B phosphopeptides, anti-phosphopeptide antibodies, and methods for cancer diagnosis and drug screening
IN Ducommun, Bernard; Monsarrat, Bernard; Prigent, Claude
PA Centre National de la Recherche Scientifique CNRS, Fr.
SO Fr. Demande, 32 pp.
CODEN: FRXXBL
DT Patent
LA French
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	FR 2856068	A1	20041217	FR 2003-7095	20030612
	FR 2856068	B1	20050819		
	CA 2528844	A1	20041223	CA 2004-2528844	20040608
	WO 2004111215	A1	20041223	WO 2004-FR1416	20040608

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,

TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

EP 1631665 A1 20060308 EP 2004-767282 20040608
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK

PRAI FR 2003-7095 A 20030612
WO 2004-FR1416 W 20040608

AB A phosphopeptide derived from human protein phosphatase CDC25B, i.e., TPVQNKRRRSpVTPPEEQQE, is disclosed. Also disclosed are polyclonal or monoclonal antibodies binding to this phosphopeptide. These antibodies may be used in diagnosis of breast cancer or in screening for antitumor agents. Thus, the site of phosphorylation of human CDC25B and its splice variants by protein kinase aurora A/STK5 was determined

RE.CNT 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 11 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:990980 CAPLUS
DN 140:40888
TI Monoclonal antibodies to Aurora A kinase and their use in the diagnosis and treatment of cancer
IN Prigent, Claude; Martin, Anne
PA Centre National De La Recherche Scientifique Cnrs, Fr.
SO Fr. Demande, 33 pp.
CODEN: FRXXBL
DT Patent
LA French
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	FR 2840905	A1	20031219	FR 2002-7212	20020612
	FR 2840905	B1	20060707		
	CA 2489214	A1	20031224	CA 2003-2489214	20030612
	WO 2003106500	A1	20031224	WO 2003-FR1772	20030612

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

	AU 2003255671	A1	20031231	AU 2003-255671	20030612
	EP 1511771	A1	20050309	EP 2003-760023	20030612

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK

	JP 2006513135	T	20060420	JP 2004-513330	20030612
	US 2007117163	A1	20070524	US 2005-517645	20050210
PRAI	FR 2002-7212	A	20020612		
	WO 2003-FR1772	W	20030612		

AB The present invention has as an aim a monoclonal antibody directed against kinase aurora-A of the mammals, its process of obtaining, as its uses within the framework of the diagnosis or the forecast of cancers, and in pharmaceutical compns. within the framework of the treatment of cancers. Monoclonal antibodies have been raised against the Aurora A kinase for use in the diagnosis, prognosis, and treatment of cancer. The

monoclonal antibody 35C1 does not inhibit Aurora A kinase.

RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 12 OF 12 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:958062 CAPLUS
DN 138:285769
TI Preparation and characterization of a human aurora-A kinase monoclonal antibody
AU Cremet, Jean Yves; Descamps, Simon; Verite, Frank; Martin, Ann; Prigent, Claude
CS Faculte de medecine, IFR 97 Genomique et Sante, CNRS - UMR 60611 Genetique et Developpement, Universite de Rennes 1, Rennes, 35043, Fr.
SO Molecular and Cellular Biochemistry (2003), 243(1&2), 123-131
CODEN: MCBIB8; ISSN: 0300-8177
PB Kluwer Academic Publishers
DT Journal
LA English
AB We have developed monoclonal antibodies against the human aurora-A serine/threonine kinase. After immunization of a mouse, a fusion was performed to obtain hybridomas that were selected because they produced Ig pos. reacting against the protein used for immunization. We isolated one particular monoclonal that we named 35C1 using a series of selective assays. The first criteria of the screen for monoclonals was an Elisa (Enzyme Linked Immunosorbant Assay) assay performed in 96-well plates against the purified recombinant histidine-tagged aurora-A. The second was a pos. Western blot against the same recombinant protein. The third criteria was a pos. western blot against an HeLa cell extract, the selected monoclonal should detect only one protein migrating at 46 kDa (kiloDalton) on SDS (Sodium Dodecyl Sulfate)-polyacrylamide gel electrophoresis. Finally, the monoclonal had to bind to duplicated centrosomes and spindle poles in human MCF7 cultured cells by indirect immunofluorescence. At this stage several monoclonals were still pos. We then increased the selectivity by searching for antibodies that were able to cross-react with the mouse aurora-A kinase both by western blot and indirect immunofluorescence. We selected and cloned the 35C1 hybridoma to produce the antibody. Further characterization of the 35C1 antibody revealed that it was able to immunoppt. the kinase, that it did not inhibit the aurora-A kinase activity and consequently could be used to measure the aurora-A kinase activity in vivo after immunopptn.

RE.CNT 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

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=> s (aurora-related kinase 1) or (hark1) or (breast-tumor-amplified kinase)
          4145 AURORA
           495 AURORAS
          4226 AURORA
                 (AURORA OR AURORAS)
       1150462 RELATED
             1 RELATEDS
       1150463 RELATED
                 (RELATED OR RELATEDS)
        299443 KINASE
         57417 KINASES
        308775 KINASE
                 (KINASE OR KINASES)
       9279975 1
             4 AURORA-RELATED KINASE 1
                 (AURORA(W)RELATED(W)KINASE(W)1)
             0 HARK1
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         79660 BREAST
           680 BREASTS
         79877 BREAST
                 (BREAST OR BREASTS)
        421181 TUMOR
        161919 TUMORS
        472108 TUMOR
                 (TUMOR OR TUMORS)
         80731 AMPLIFIED
        299443 KINASE
         57417 KINASES
        308775 KINASE
                 (KINASE OR KINASES)
             5 BREAST-TUMOR-AMPLIFIED KINASE
                 (BREAST(W)TUMOR(W)AMPLIFIED(W)KINASE)
L6           9 (AURORA-RELATED KINASE 1) OR (HARK1) OR (BREAST-TUMOR-AMPLIFIED
               KINASE)

=> s l6 and monoclonal
        148722 MONOCLONAL
           543 MONOCLONALS
        148789 MONOCLONAL
                 (MONOCLONAL OR MONOCLONALS)
L7           1 L6 AND MONOCLONAL

=> d l7 bib abs 1

L7   ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2007 ACS on STN
AN   2002:555652  CAPLUS
DN   137:120714
TI   Human cDNA sequences and their encoded proteins and diagnostic and
     therapeutic uses
IN   Edinger, Shlomit; MacDougall, John R.; Millet, Isabelle; Ellerman, Karen;
     Stone, David J.; Gerlach, Valerie; Grosse, William M.; Alsobrook, John P.,
     II; Lepley, Denise M.; Rieger, Daniel; Burgess, Catherine E.; Casman,
     Stacie J.; Spytek, Kimberly A.; Boldog, Ferenc L.; Li, Li; Padigaru,
     Muralindhara; Mishra, Vishnu; Patturajan, Meera; Shenoy, Suresh; Rastelli,
     Luca; Tchernev, Velizar T.; Vernet, Corine A. M.; Zerhusen, Bryan D.;
     Malyankar, Uriel M.; Guo, Xiyojia; Miller, Charles E.; Gangolli, Esha A.
PA   Curagen Corporation, USA
SO   PCT Int. Appl., 353 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 175
     PATENT NO.          KIND   DATE        APPLICATION NO.         DATE
     ---------------     ----   --------    --------------------    --------
PI   WO 2002057450        A2    20020725    WO 2001-US48922         20011129
     WO 2002057450        A3    20030717
        W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
            CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
            GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
            LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
            PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA,
            UG, US, UZ, VN, YU, ZA, ZM, ZW
        RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
            KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB,
            GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA,
            GN, GQ, GW, ML, MR, NE, SN, TD, TG
     AU 2002246696        A1    20020730    AU 2002-246696          20011129
     US 2004029116        A1    20040212    US 2002-87684           20020301
     US 7109000           B2    20060919
     US 2004029222        A1    20040212    US 2002-218779          20020814
     AU 2005200106        A1    20050210    AU 2005-200106          20050112
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	AU 2006201467	A1	20060504	AU 2006-201467	20060407
PRAI	US 2000-253834P	P	20001129		
	US 2000-250926P	P	20001130		
	US 2001-264180P	P	20010125		
	US 2001-313656P	P	20010820		
	US 2001-327456P	P	20011005		
	US 2001-995514	A	20011128		
	AU 2000-37360	A3	20000309		
	AU 2000-78680	A3	20001006		
	US 2001-274194P	P	20010308		
	WO 2001-US48922	W	20011129		

AB Disclosed herein are 12 cDNA sequences that encode novel human polypeptides that are members of the following protein families: transmembrane receptor UNC5H2-like, tyrosine phosphatase precursor-like, glomerular mesangial cell receptor protein tyrosine phosphatase precursor-like, Drosophila pecanex-like, Aurora-related kinase 1-like, 26S protease regulatory subunit 4-like, mitsugumin29-like, Wnt-15-like, Wnt-14-like, β-adrenergic receptor kinase-like, α-mannosidase-like, C1q-related factor-like, and plexin 1-like. Also disclosed are polypeptides encoded by these nucleic acid sequences, and antibodies, which immunospecifically-bind to the polypeptide, as well as derivs., variants, mutants, or fragments of the aforementioned polypeptide, polynucleotide, or antibody. The invention further discloses therapeutic, diagnostic and research methods for diagnosis, treatment, and prevention of disorders involving any one of these novel human nucleic acids and proteins.